Contents

The President's Page — The Physician and Community Leadership ..... Page 755
By Russell L. Deter, M.D., El Paso

Lead Exposure — A Common Cause of Disease ..... Page 757
By R. P. Watterson, M.D., Scottsdale, Ariz.

Dr. Charles J. Newcomb of Tucson Heads Southwest OB and GYN Society ..... Page 761

District One of Texas Medical Association to Meet in Pecos ..... Page 762

Southwest OB and GYN Society Annual Meeting in Palm Springs, Calif. Report in Pictures ..... Page 763

Clinical Experiences With A New Long Acting Vitamin B_{12} ..... Page 767
By H. E. Kimble, M.D., Chicago

Clinical Pathological Conference, R. E. Thomason General Hospital, El Paso ..... Page 771
Frederick P. Bornstein, M.D., Editor
Presentation of Case by Solomon Heller, M.D.

Coming Meetings

Medical Society of the United States and Mexico, Annual Meeting, Hotel Valley Ho, Phoenix, Dec. 2-6, 1959.

American Medical Association, 13th Clinical Meeting, Dallas Memorial Auditorium, Dallas, Dec. 1-4, 1959.

University of Colorado School of Medicine, General Practice Review, Denver, Jan. 10-16, 1960.

American Diabetes Association, Postgraduate Course, Ambassador Hotel, Los Angeles, Jan. 20-22, 1960.

International Medical Assembly of Southwest Texas, 24th Annual Meeting, Hilton Hotel, San Antonio, Jan. 25-27, 1960.

Texas District One Medical Association, Annual Meeting, Pecos County Club, Pecos, Tex., Feb. 5, 1960. Chairman, Dr. Harold Lindley, 206 S. Oak St., Pecos.

New Mexico Medical Society, Annual Meeting, Western Skies Hotel, Albuquerque, May 11-13, 1960.

Southwest Obstetrical and Gynecological Society, Annual Meeting, Las Vegas, Nev., Nov. 7-8, 1960.

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References:

1. Carpenter, E. B.: Southern M. J. 51:627, 1958. 2. Forsyth, H. F.: J.A.M.A. 167:163, 1958. 3. O'Doherty, D. S., and Shields, C. D.: J.A.M.A. 167:160, 1958. 4. Park, H. W.: J.A.M.A. 167:168, 1958. 5. Plumb, C. S.: Journal-Lancet 78:531, 1958.

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SOUTHWESTERN MEDICINE

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THE PRESIDENT'S PAGE

Dr. Deter

The Physician and Community Leadership

By RUSSELL L. DETER, M.D., *El Paso*

In my first "President's Page" I wish to express my appreciation for the honor you have bestowed upon me in electing me President of the Southwestern Medical Association — an association which, in all its history of 41 years, has been characterized by the neighborliness of its membership.

Our meetings have been outstanding in presentation of its scientific speakers, but more than that—these meetings have afforded us with an opportunity once a year to enjoy the company of our friends and their families, much in keeping with the old tradition of the southwest camp meetings.

My purpose this year, through these pages, will be to urge you to participate more actively in your county medical society affairs and your community affairs, and to keep you posted on changes in national politics and legislation, as it pertains to the medical profession.

Big Problem

Today, one of our big problems in medicine is to find methods by which we can stir up the mass inertia of a vast segment of the medical population —not only regarding problems which concern medicine specifically, but problems which concern the nation as a whole. It is extremely difficult for 30 members of a 300-member county society to conduct the business of that society to the satisfaction of a majority. This probably is one explanation for dissatisfaction with some of the decisions of your local county medical society. The question is—WERE YOU THERE??. Quoting from an article by Dr. Roger L. Buck, M.D., in "The Trustee"—the journal for Governing Hospital Boards—

"I will not attempt to discuss the reasons, but physicians today seldom assume the community

leadership roles of their predecessors. A family physician who was, at the same time, a leader in the community, commanded prestige from his patients.

Active Concern

He was respected for his active concern in the patient, and in the patient's community. There was less chance of his having to labor under the stigma of a label of commercialism imposed by a suspicious or poorly informed public. Examination of the problems of the widening gap of the physician-patient relationship demonstrates that forces are involved that necessitate assistance from beyond the ranks of the medical profession. Solutions of these problems will be a task for research and other collaboration between social science and medicine.

Let us, therefore, become involved with our entire community problems, and not be so intimately concerned with our own private little rat race.

I would like to avail myself of this opportunity to wish you a Very Merry Christmas and a Happy and Prosperous New Year.

Synthetic Drug Successful In Treating Skin Infections

A new, orally administered antibacterial drug has been highly successful during the past few months in treating almost ninety per cent of patients with a wide variety of skin infections at the department of dermatology of the University of Cincinnati's College of Medicine.

Most of these infections have been due to staphylococci, the bacteria primarily responsible for the increase of drug-resistant infections in hospitals, and some have been resistant to other antibiotic agents. Dr. Alfred L. Weiner, associate professor of dermatology at the college, reported on treatment of skin infections with the new drug, Altafur, at a conference on the management of chronic dermatoses at the college here today.

Significant Results

These results are significant, Dr. Weiner said, because "the medical profession may in the future have to rely more and more on synthetic, man-made antibacterial drugs rather than on the once remarkably effective antibiotics, derived from nature, because of the emergence of increasing numbers of resistant strains of bacteria and the increasing number and hazard of reactions to them."

Altafur, whose generic name is furaltadone, is the newest of the synthetically derived nitrofurans, distinct from the antibiotics and the sulfa drugs. It has been effective, Dr. Weiner reported, in treating 35 out of 40 patients with boils, carbuncles, bacterial skin infections accompanying eczema, acne, cellulitis, and other infections caused by staphylococci. Besides the forty patients whose progress has been followed carefully, an additional 22 have been treated in the short time his investigation has been underway, and initial study indicates that results will probably parallel those obtained with the first group of patients.

Earlier Studies

Earlier sensitivity studies with Altafur showed that it was inhibitory to 449 of 476 cultures of staphylococcus aureus, including 220 of the so-called phage-type 80/81 organisms, the ones causing the greatest problems of resistance to antibiotics in hospitals.

The incidence of side reactions to the new drug was low, Dr. Weiner said, with five instances of nausea and vomiting, one of dizziness, and an unusual reaction of rash, fast pulse and difficult breathing in a few patients who had taken alcohol during drug therapy. The latter reaction resembled that seen with Antabuse, a drug used to combat alcoholism. Eaton Laboratories, developers and producers of the nitrofurans, have cautioned physicians that alcohol must not be ingested during and for at least seven days after Altafur therapy.

Resistant Strains

Dr. Weiner pointed out that since resistant strains of bacteria evidently emerge chiefly in hospitals, reevaluation and improvement of current hospital aseptic and housekeeping techniques is necessary.

Minor skin infections caused by staphylococci can be treated simply with topical drugs. Systemic treatment should be reserved for those that are extensive, unresponsive or resistant. "And it is in this area," Dr. Weiner said, "that the chemotherapeutic agents will have increasing application in the future."

Lead Exposure — A Common Cause of Disease

By R. P. Watterson, M. D., *Scottsdale, Ariz.*

The lethal and crippling role of lead is played in two parts. One is the activation of hyaluronidase (the spreading factor enzyme that liquefies connective tissue) and the second is the blocking of heme formation.

Heme is the iron porphyrin chelate that is the basic structure of all enzymes which transport, utilize and store oxygen. These enzymes include all of the oxidases, catalase, hemoglobin, myoglobin, slater factor, tyrosinase and the cytochrome groups.

Lead blocks the chelation of iron with the porphyrin ring thereby limiting the heme available for synthesis of porphyrinic enzymes. If porphyrin synthesis and absorption proceeds normally which this chelation mechanism is blocked the porphyrin fractions accumulate with resultant excretion. This phase is recognized as porphyria. The associated dysfunction which is due to a low titer of porphyrinic enzymes disturbs all phases of intermediary metabolism and total function.

Adequate attention has not been drawn to the fact that many substances inhibit porphyrin synthesis which if coexistant with lead activity would prevent porphyrin accumulation and excretion, meanwhile, markedly lowering the titer of porphyrinic enzymes. The technical difficulties in bioassay of these enzymes has prevented adequate study.

Water Retention

Hyaluronidase activity in liquefaction of connective tissue and cartilage is well documented. The fact that hyalinization of these structures results in water retention due to release of mucoids, mucopolysaccharides, etc., with resulting symptoms of anoxia, electrolyte disturbance and defensive changes in intermediary metabolism, has not been assimilated into the picture of pathologic physiology by the majority of medical minds.

Without lead, hyaluronidase is inert. With this point in mind, the necessity for reducing lead exposure, promoting deposition of lead by simulating calcification of bone and enhancing excretion of lead becomes apparent. At the clinical level, control of the lead factor and adequate supply of essential nutrients for maintenance of structural and functional stability can be merged with antibiotic and chemotherapeutic attacks on hyaluronidase-producing bacteria. This more comprehensive program also relieves the metabolic pressures attendant with suppression of heme formation which exist while lead remains in solution in tissue fluids.

Pertinent Instructions

For both prevention and treatment recommendations to patients, the following instructions are pertinent.

1. Reduce lead exposure by avoiding smog, car exhausts, lead-contaminated foods, chemical gases, excessive handling of paint and lead-containing objects.
2. Eliminate magnesium-low foods (white sugar, Karo and white flour). Since magnesium is required for the combustion of sugar and starch, and inhibition of hyaluronidase, a magnesium-low diet results in mobilization of magnesium from bone with decalcification and release of lead from bone.
3. Avoid symptomatic steroid therapy to prevent decalcification with its attendant mobilization of lead.
4. Reduce stress to a minimum. Excessive endogenous secretion and the attendant catabolic processes are prevented.
5. Take additional magnesium (in absorbable form), vitamin C. bioflavinoids, paba, vitamins A, D and E and calcium pantothenate when the signs and symptoms of hyaluronidase activity are evident. These nutritional factors stabilize tissue and inhibit hyaluronidase. Hyalex (Miller Pharmacal Company) combines these factors.
6. Take supplementary HC1 with meals to assist assimilation. Without adequate HC1 in stomach juice, calcium is not assimilated.
7. Versenate (Riker) can be used to increase

lead excretion if chronic lead exposure is unavoidable, or serum lead levels remain elevated.

8. Utilize testosterone or estrogen therapy or both, as indicated, for anabolic stimulus to ossification and deposition of lead, thus clearing or lowering serum lead levels.

Excerpts

For convenience in reference, the following excerpts from the literature are presented.

The metal activator of hyaluronidase is **lead.** (Blackburn, C.R.B., J. Biol. Chem. v. 278, p. 855-1949.)

"If the blockage of the reticulo-endothelial system takes place at the same time as the lead poisoning then the acute anaemia appears more rapidly, the basophilic granulation more slowly, and the level of serum iron seems to remain unchanged at its normal value. Quantitative examination of the iron in organs rich in reticulum tissue reveals decidedly lower values than in a corresponding rabbit with simple lead poisoning. In the spleen, on the other hand, we often observed normal iron values . . .

"These experiments lead us to conclude that lead poisoning causes an inhibition of the utilization of iron in the synthesis of haemoglobin in the erythroblast. This causes the formation of a blood pigment minus iron—a porphyrin of type III." (Vannotti, A., Porphyrins, p. 130 and 131, 1954).

A low order of hemoglobin production, with porphyrin production proceeding at the normal levels, can potentially result in what would be regarded as excessive excretion of porphyrins. This is the situation in lead poisoning wherein utilization of iron and the synthesis of hemoglobin appear to be impaired. (Drabkin, D. L., Physiol. Rev., v. 31, p. 411—1951).

"Recent work has demonstrated that Coproporphyrin III formed during lead intoxication is not derived from haemoglobin degradation but from disordered haem synthesis (Grinstein, Wikoff, de Mello and Watson)" . . . (Vannotti, A., Porphyrins, p. 32—1954).

In "Review of Physiological Chemistry" by H. A. Harper, 6th Ed., p. 65, there is a diagram showing that Coproporphyrin III is an immediate precursor of Heme—which is in turn utilized to form the oxidative enzyme systems, whether it be hemoglobin, myoglobin, catalase or the cytochromes. Therefore, all the enzymes in the oxygen transport, storage and utilization are basically porphyrin derived compounds.

"Coproporphyrin III is excreted in excessive quantities as the result of (1) exposure to certain toxic chemicals and heavy metals (e.g., lead)" . . . (Cantarow, A., and Schepartz, B., Biochemistry, p. 596—1954).

Porphyrins are high in liver during pernicious anemia, heart disease, lead poisoning and lymphogranuloma, etc. (Noering, M.Z. ges inn. Med., v. 4, p. 182—1949).

In thalassemia (Cooleys anemia) and in lead poisoning, red blood cells deficient in hemoglobin are found despite the abundance of iron granules within the normoblasts. (Kaplan, E. et al, Blood, v. 9, p. 202—1954).

". . . Industrial poisoning with lead is more common through the respiratory tract than by ingestion. Lead fumes and dusts are readily absorbed from the lungs and pass directly into the general circulation more completely than through the gastrointestinal tract . . . Thus, lead demonstrates some of the important differences between pulmonary and intestinal absorption." (Sodeman, Wm. A., Pathologic Physiology: Mechanisms of Disease, p. 755—1950).

"The accumulation of excessive amounts of lead in the body, leading to plumbism, results from the absorption of the metal from the gastrointestinal tract, the respiratory epithelium or through the skin . . . In industry, the respiratory and cutaneous routes of absorption are more important than the oral . . .

". . . The addition of tetra-ethyl lead to gasoline constitutes an important source of skin penetration leading to plumbism. In industry, the inhalation of lead fumes is the chief portal of entry and is the most important route leading to lead poisoning. Lead that is inhaled is much more toxic than lead which is ingested.

". . . When large quantities of lead are inhaled or absorbed, excretion does not keep pace and storage occurs . . . Immediately after absorption, the greatest amount of lead appears in the spleen, liver and kidneys. After a few days, it collects entirely in the bones . . . Not all the lead deposited in bone is excreted when it is liberated. It is repeatedly dissolved and redeposited along the epiphyseal lines of growth, especially in child-

ren . . . Early signs of lead poisoning include a mild polycythemia; polychromatophilia; increase in platelets and reticulocytes. The reticulocyte is an early sensitive sign of lead intoxication. Later, there are evidences of bone marrow depression including anemia; increase in basophilic cells; increase in mononuclears; anisocytosis; poikilocytosis; nucleated red blood cells and thrombocytopenia." (Hyman, H. T., An Integrated Practice of Medicine, p. 762 and 763—1946).

Lead in bones occurs especially in fat cells of bone marrow and x-rays do not show it. It deposits in all bone surfaces over which blood passes. (U. S. Public Health Reports, No. 58, p. 209-216—1943).

Note well that porphyrins are probably found in all living cells — including all human cells — although they are concentrated in the bone marrow (Everett Med. Biochem., p. 540 and Granick, S., Gilder, H., Advances in Enzymology, v. 7, p. 306—1947) and are associated especially with nucleated erythrocytes. (Larsen, E. G. et al, Fed. Proc. v. 12, p. 236 (No. 777)—1953).

It should be emphasized that all known vitamins and twelve or more minerals are essential in normal heme synthesis. The same is true of chlorophyll synthesis, in which connection we are reminded that the formation of the porphyrin ring by the chlorella plant follows almost an identical pattern, whether chlorophyll is the end-product or heme is the ultimate goal. (Della Rosa, R. J. et al, J. Biol. Chem., v. 202, p. 771—1953; Granick, S. et al, Ibid v. 202, p. 793-813—1953).

In acute porphyria hormonal relationship and allergy factors are involved. (Dorken, H., J.A.M.A., v. 152, p. 1282—1953 Abs.).

Diamine oxidase functioning with the dehydrogenase systems, destroys histamine. Diamine oxidase is formed from heme. Its only variance from other porphyrinic enzymes being its prosthetic side chains. It is logical to conclude that any major suppression of heme synthesis woud result in a decreased titer of diamine oxidase. The clinical manifestation of this low titer would be the signs of allergic disorder. (Cantarow, A., & Schepartz, B., Biochemistry, p. 274, 358-368—1954; Martin, C. J., Biological Antagonism, p. 44-47—1951).

Rheumatic carditis is associated with considerable increase in hyaluronidase and histimine. Clinical improvement parallels lowering the levels of both. (Stepanyan, E. P. and Perchikova, O. E., Clin. Med. v. 35, #5, p. 129—1957).

Catalase (a porphyrin-derived compound, and an oxidative enzyme) in liver is correlated inversely with severity of disease and is particularly low in infectious hepatitis. (Dale, B.G., Am. J. Med. Sci., v. 226, p. 42—1953).

In cancer, liver catalase is greatly diminished through interference with synthesis of hematoporphyrin ring. (Greenstein, J.P., J. Natl. Cancer Inst., v. 3, p. 397—1943).

Hyaluronic acid is found in connective tissues, synovial fluids, cartilage, vitreous humor, the cornea, umbilical cord, . . . and in many bacteria. It is concerned with rheumatism and the fluid transfer of tissues. (Gross, J., J. Biol. Chem., v. 172, p. 511—1948).

The hyaluronic acid of the intervertebral discs is liberated and decomposed by hyaluronidase. (McClure, C. et al, Science v. 230, p. 189—1954).

"Hyaluronic acid is a mucopolysaccharide found in animal tissues, whose function appears to be that of binding water in interstitial spaces, holding cells together in a jelly-like matrix and serving as a lubricant or shockabsorber in joints . . . long chain compounds of hyaluronic acid plus proteins give tensile strength and elasticity to connective tissues. In synovial fluids the viscosity is due largely to this hyaluronic acid-protein complex. The hyaluronidase (enzyme) breaks down this viscosity to watery consistency." (Arts and Osman, Biochem. of Glucuronic Acid. p. 18-20).

Injection of hyaluronidase into the joints of living animals causes "hydrolysis of hyaluronic acid (content) with decrease in viscosity." (Regan, C. and De Lamater, A., Proc. Soc. Exp. Biol. Med. v. 50, p. 349—1942).

The arthritic condition affects the collagen tissues of the body (Frick, P.G., Blood, v. 10, p. 691) and the muco-protein of cartilage. (Shubert, M. and Shatten, J., Ann. Rheum. Dis., Proc. v. 14, p. 95—1955).

The hyaluronic acid of the intervertebral discs is liberated and decomposed by hyaluronidase. (McClure, C. et al, Science v. 119, p. 189—1954).

In gout, a lack of circulating hyaluronic acid tends to cause retention of uric acid. (Allen, G.F. et al, Am. J. Med. Sci., v. 230, p. 30—1955).

Rheumatic carditis is associated with considerable increase in hyaluronidase and histamine. Clinical improvement parallels lowering the levels of both. (Stepanyan, E.P. and Perchikova, O.E., Clin. Med. v. 35, no. 5, p. 129—1957).

Hyaluronidase and deoxycorticosterone increase permeability of "ground substances" by depolymerizing hyaluronic acid. In vitro, hyaluronidase destroys the "plasma clearing factor," indicating the latter contains a mucopolysaccharide. (Seifter, J. and Baeder, D.H., Proc. Soc. Exp. Biol. Med. v. 86, p. 709—1954).

Hyaluronidase is inhibited by magnesium as blood serum of magnesium is raised from 0.05 to 0.1 mEq. (Domonkos, J. et al, Acta Physiol. Scand. Sci. Hungary, v. 6, p. 11—1954).

Magnesium deficiency may cause excessive calcification of the bones of the kidneys, heart and arteries, along with dermatitis, vasodilation and convulsions (including tetany). Much of the magnesium intake is lost through the bowel. (Everett's Med. Biochem. p. 591-593—1946).

Release of excess histamine into the blood stream may be prevented by magnesium. (McIntine, F.C. et al, Am. J. Physiol. v. 159, p. 332).

Vitamin A levels of the blood are usually low in arthritic conditions. The same is true of the Vitamin B complex, as well as of Vitamin D and E. (Sachler, Acta. Med. Scand. v. 110, p. 285—1942).

The toxicity of lead is materially lessened by adequate Vitamin C. (Frommel, E. and Loufti, M., Arch. Sci. Phys. Nat. v. 26, p. 149—1944).

In the biosynthesis of collagen (connective tissue) "Procollagen" has been found to be an intermediate substance, the formation of which depends directly upon adequate Vitamin C. (Orckhovitch and Shipkiter, V.O., Science, v. 127, p. 1371—1958).

Tocopherols inhibit hyaluronidase both in vitro and vivo, so Vitamin E may be useful in the treatment of collagen disease. (Grifa, P. Minerva Med. v. 43-1, p. 270—1952).

Capillary permeability increases in Vitamin E deficiency, with resulting accumulation of unsaturated fatty acids, and exudation diathesis. (Dam, H., Proc. Soc. Exp. Biol. Med. v. 52, p. 285—1943).

In experiments on rats using DHT (dihydrotachysterol) and NaH_2PO_4, Selye precipitated widespread calcification in the aorta, and in the heart there was calcification of the Monckeberg type in the coronary vessels. In addition there were many areas of myocardial necrosis and thrombi developed adjacent to the areas of necrotic tissue. These thrombi and necrosed areas were identical to that seen in human hearts. The necrosed areas and calcification were completely prevented by $MgCl_2$ and almost completely prevented by KCl. (Selye, H., Am. Heart J., v. 55, #6, p. 805-809—1958).

Magnesium activates more enzyme systems, including those of the glycolytic series and the Krebs cycle, than any other mineral. (J. Am. Chem. Soc. v. 76, p. 2279—1954; J. Biol. Chem. v. 216, p. 215—1955).

Magnesium inhibits the "spreading factor," which destroys connective tissues, known as hyaluronidase. (Freeman et al, Proc. Soc. Exp. Biol. Med. v. 70, p. 524—1949).

Adrenal cortex activity is intimately associated with pantothenate supply and the latter vitamin is a part of "co-enzyme A" which plays an essential role in the metabolism of carbohydrates, fats, steroid hormones and cholesterol. (Nut. Revs. v. 6, p. 201—1948; J. Biol. Chem. v. 200, p. 839—1953; Hurley, L. C. et al, J. Nut. v. 54, p. 403—1954).

Deficiency of so-called Vitamin P (bioflavonoids) predisposes to degenerative and infectious disease, as "Vitamin P." inhibits hyaluronidase. (Levitan, B. A., Am. J. Physiol. v. 157, p. 422—1949).

Hesperidin seems to be a natural inhibitor of hyaluronidase. (Chem. Eng. News, v. 30, p. 4391—Edit.).

Summary

The roles of lead in blocking heme formation and activation of hyaluronidase have been presented. The far-reaching effects of these actions can be projected by those familiar with physiology and pathology.

Physiologic measures are suggested for the patient showing signs of connective tissue disturbance. Laboratory diagnosis of lead accumulation remains unsatisfactory.

Post Office Box 1027

MEETINGS

Dr. Newcomb

Dr. Charles J. Newcomb of Tucson Heads Southwest OB-GYN Society

Dr. Charles J. Newcomb of Tucson was elected President of the Southwest Obstetrical and Gynecological Society at its ninth annual meeting in Palm Springs, Calif., November 1-3.

Other officers elected at the meeting were Dr. John Wanless, San Diego, President-elect; Dr. Robert O'Donnell, Las Vegas, Nev., Vice-President; Dr. Raymond Jennett, Phoenix, Treasurer; and Dr. Zeph B. Campbell, Phoenix, Secretary. Doctors Jennett and Campbell were both re-elected. Dr. Donovan Johnson of Santa Ana, Calif., was the retiring President.

New members of Council Group "B" are Dr. Carlos Craig, Phoenix, Dr. Ray Young, Santa Fe, Dr. Paul Peterson, Fullerton, Calif., Dr. Paul O. Wiig, Reno; Dr. Charles T. Franklin, La Mesa, Calif; and Dr. Bernard Hark, La Jolla, Calif.

Members of Council Group "A", who continue in office for another year, are Dr. James Ravenscraft, San Diego, Dr. Gray Carpenter, El Paso, Dr. Louis McRae, Albuquerque, and Dr. Herman Rhu, Tucson.

Meeting In Las Vegas

The 1960 meeting will be held in Las Vegas, Nev., with November 7 and 8, 1960, having been selected as tentative dates.

Speakers who addressed the scientific sessions were as follows:

Dr. Ralph C. Benson, Professor and Chairman of the Department of Obstetrics and Gynecology, University of Oregon Medical School, Portland, Ore.; Dr. Russell R. de Alvarez, Professor and Executive Officer of the Department of Obstetrics and Gynecology, University of Washington School of Medicine, Seattle.

Dr. Bernard J. Hanley, Clinical Professor of Obstetrics and Gynecology and Chairman of the Department of Obstetrics, University of Southern California Medical School, Los Angeles; Dr. Charles E. McLennan, Professor and Chairman

of the Department of Obstetrics and Gynecology, Stanford University School of Medicine, San Francisco.

Dr. Daniel G. Morton, Professor and Chairman of the Department of Obstetrics and Gynecology, University of California, Los Angeles; Dr. Ernest W. Page, Professor and Chairman of the Department of Obstetrics and Gynecology, University of California, San Francisco; and Dr. Ralph R. Reis, Professor of Obstetrics and Gynecology, Northwestern University Medical School and Editor of *Obstetrics and Gynecology,* Chicago.

Born In Wisconsin

Dr. Newcomb was born in Waupun, Wis., and received his B.A. from the University of Wisconsin and his M.D. from the University of Wisconsin School of Medicine. He interned in St. Louis City Hospital from 1932 to 1933 and then took a year's surgical residency in the La Crosse Lutheran Hospital in Wisconsin. He was at the Lying-in Hospital in Chicago for five years for his Obstetrical and Gynecological residency.

He began the private practice of medicine in 1939 in Milwaukee, where for four years he was Clinical Instructor of Obstetrics and Gynecology at Marquette University School of Medicine. He served in the Navy for four years, with one year in the South Pacific, and emerged from the service with the rank of Lieutenant Commander. In 1946 he opened his office in Tucson.

Dr. Newcomb is President of the Tucson Obstetrical and Gynecological Society, a Diplomate of the American Board of Obstetrics and Gynecology, a Fellow in the American College of Obstetrics and Gynecology and a member of the Central Association of Obstetrics and Gynecology. He and Mrs. Newcomb have a daughter and two sons. He is a member of St. Phillips of the Hills Episcopal Church.

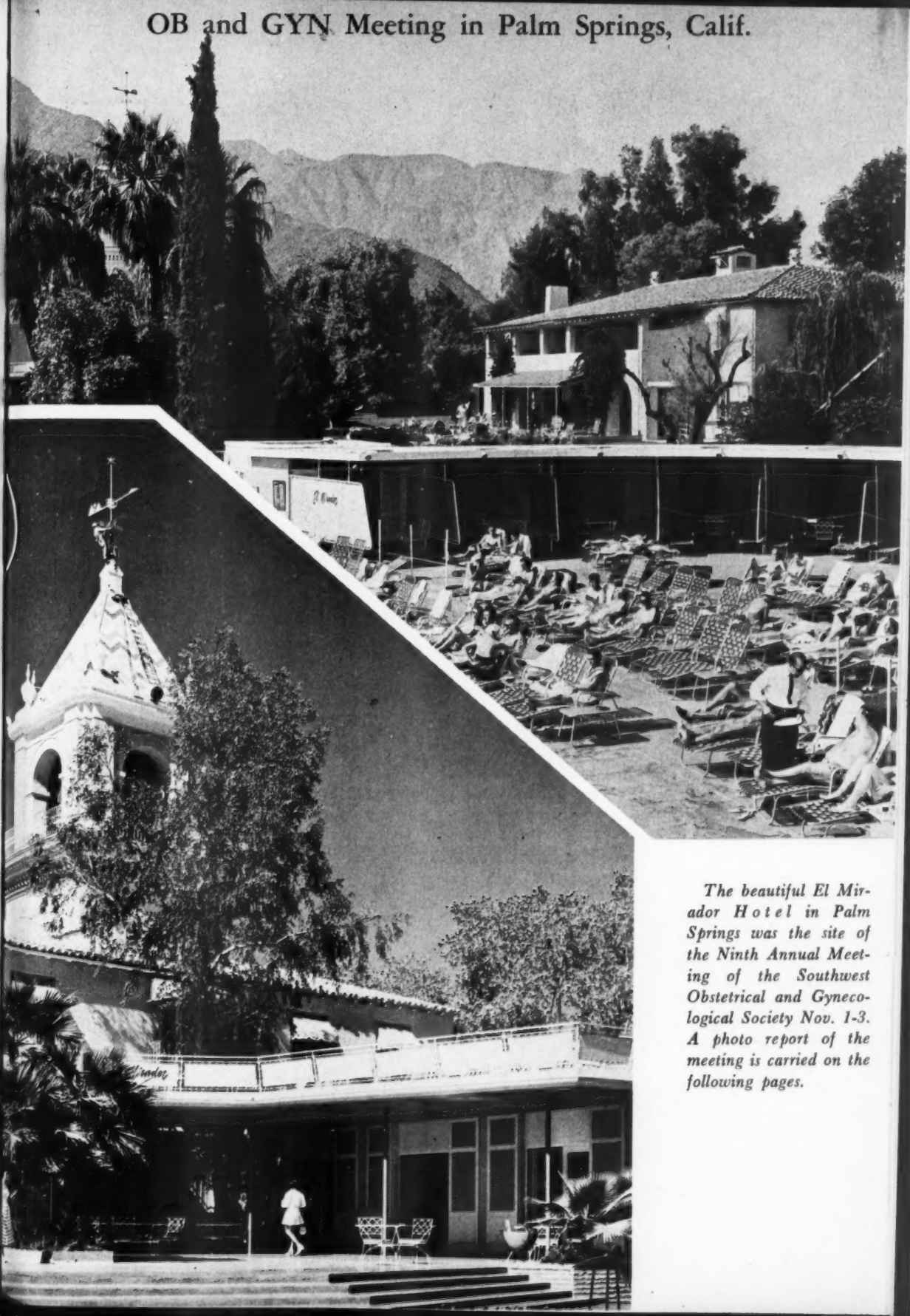
Texas Medical President To Speak to District One

Dr. Franklin W. Yeager, president of the Texas Medical Association, is slated to be the principal speaker at the District One meeting of the Texas Medical Association in Pecos, Texas, February 5, 1960.

Other speakers on the tentative program are Dr. Ira A. Budwig, Dr. E. Samuel Crossett, Dr. John M. Verosky, Dr. C. M. Stanfill, Dr. Jack C. Postlewaite, Dr. Nathan M. Kleban, Dr. William G. Smith, Dr. Albert H. Unger, Dr. Louis W. Breck, and Dr. Frederick P. Bornstein, all of El Paso.

Officers of District One are Dr. W. E. Lockhart, Alpine, president; Dr. W. H. McClure, Kermit, vice-president; and Dr. Gordon L. Black, El Paso, secretary-treasurer.

OB and GYN Meeting in Palm Springs, Calif.



The beautiful El Mirador Hotel in Palm Springs was the site of the Ninth Annual Meeting of the Southwest Obstetrical and Gynecological Society Nov. 1-3. A photo report of the meeting is carried on the following pages.

Seated in the El Mirador lobby during a break in the scientific sessions were, left to right, Dr. Ralph A. Reis, guest speaker; Dr. Bernard J. Hanley, speaker; Dr. Ray C. Bitterlich, Albuquerque; Dr. Ralph C. Benson, speaker; Dr. Daniel G. Morton, speaker; and Dr. Charles E. McLennan, speaker. In the foreground, back to camera, is Dr. Ernest A. Page, Speaker.

Left to right, Dr. Basil W. Maloney, Lemon Grove, Calif.; Dr. Walter M. Ballard, San Diego; Dr. Charles E. Weber, La Jolla, Calif.; and Dr. Robert D. Cloyes, La Mesa, Calif.

Left to right, Dr. Donovan Johnson, Santa Ana, Calif., retiring president; Dr. Raymond J. Jennett, Phoenix, re-elected treasurer; Dr. Robert P. O'Donnell, Las Vegas, new vice-president; Dr. John F. Wanless, San Diego, president-elect; and Dr. Zeph B. Campbell, Phoenix, re-elected secretary.

Clockwise, from upper left . . .

Left to right, Mrs. Carlos Craig, Mrs. Robert E. Jones, Mrs. Charles E. Van Epps, and Mrs. Preston T. Brown, all of Phoenix.

Mrs. Charles I. Newcomb, left, wife of the Society's new president; and Mrs. George Fraser, both of Tucson, enjoy the Palm Springs sunshine.

Enjoying breakfast are, Mrs. Raymond J. Jennett, Phoenix, wife of the secretary-treasurer; Mrs. Ernest W. Page, wife of one of the speakers; Mrs. Jesse A. Rust, Jr., San Diego, whose husband is a past president of the Society; Mrs. Bernard J. Hanley, wife of a guest speaker; and Mrs. John Wanless, San Diego, whose husband was elected president-elect.

Left to right, Dr. John A. Jones, Phoenix; Mrs. Jones; Mrs. L. E. Kron, Phoenix; and her husband, Dr. Kron.

Dr. A. H. Thatcher, Oceanside, Calif., visits with Miss Barbara Deisher, Oceanside, and Mrs. Charles Weber, La Jolla, Calif.

Dr. Martin Cohen, Yuma, Ariz., left, and Dr. H. S. Carstensen, Tucson.

Dr. Paul O. Wüg, Reno, left, and Dr. Rudolf W. Hack, San Bernardino, Calif.

Dr. Juan Araujo, Anaheim, Calif., left, and Dr. Stanley Blake, Mesa, Ariz.

Dr. K. Ross Tucker, left, and Dr. Gustave E. Ledfors, both of Riverside, Calif.

Clinical Experiences With A New Long Acting Vitamin B_{12}

By H. E. KIMBLE, M.D., *Chicago*

Introduction

This paper is concerned with a discussion of Vitamin B_{12} (Cyanocobalamin) and with Cyanocobalamin Zinc Tannate, a repository form of Vitamin B_{12} known as DEPINAR®*.

Vitamin B_{12} is found in many of the foods we eat, mainly beef and pork, as well as in the animal organs such as the liver which contains a high concentration of this vitamin. Aside from these natural sources, Cyanocobalamin is manufactured in large amounts commercially. This supply is needed to augment our natural sources.

Vitamin B_{12} can be taken into the body by oral or parenteral administration. When taken orally it is difficult to determine how much or how little is absorbed. This is due to limited absorptive capacity under normal conditions which may be accentuated under certain pathologic conditions.

Readily Absorbed

When given parenterally in water or saline, Vitamin B_{12} is readily absorbed from the tissues directly into the blood stream. This brings about a rapid increase in the amount of the vitamin, but due to its rapid elimination in the urine, the amount of Vitamin B_{12} in the blood serum quickly returns to or near its previous level.

Experimentally most of the injected Vitamin B_{12} may be recovered in the urine within 16 hours.[1] It is quite obvious that when such a rapid rise and fall of Vitamin B_{12} in the blood serum takes place in such a relative short period of time, the various tissues and organs of the body do not apparently have sufficient time to store up any therapeutic amount of Vitamin B_{12}. Nearly all of the body tissues and organs will build up a reserve supply of Vitamin B_{12} if it is available.

Below Normal Levels

Vitamin B_{12} levels may be depleted below normal levels under certain known conditions.[2,3,4] In addition, there may be other conditions which are associated with sub optimal levels of the vitamin as suggested by Thompson and Hecht[1] and others. When this situation continues a "B_{12} starved" or relative Vitamin B_{12} deficiency condition can be said to exist. The prevention as well as treatment of "B_{12} starved" tissues and organs should be brought about most effectively by the administration of Vitamin B_{12} in a preparation with sufficient capacity for absorption control.

Recently there has been developed such a preparation of Vitamin B_{12} which in animal and human studies has shown an ideal repository behavior, in that it has a slow absorption as well as insignificant urinary excretion, even after large doses. It is so efficient that when a 500 mcg. dose is given parenterally, satisfactory serum levels are present for 28 days or longer in man.[1] As for its excretion no Vitamin B_{12} was found in the urine during the first 24 hours and then only a trace after that.[1] This repository or long acting B_{12} preparation is known as Cyanocobalamin Zinc Tannate.

Uses

Vitamin B_{12} has been used successfully in the treatment of pernicious anaemia, tropical and non-tropical sprue and also in the macrocytic and megaloblastic anaemias. It has been used with limited success in numerous other diseases and syndromes such as liver diseases,[5] alcoholism,[6] Bell's Palsy,[7] diabetic neuropathy and retinopathy,[8] herpes zoster,[9] radiculitis,[7] multiple sclerosis,[7] and

*Depinar®, (Cyanocobalamin Zinc Tannate) for these studies was generously supplied by Dr. R. A. Hecht, Armour Pharmaceutical Company, Kankakee, Illinois.

neurodermititis,[5] just to mention a few. It is felt that the limited success reported in most of these conditions is explained by the fact that oral and non-repository preparations of Vitamin B_{12} were used. Preliminary studies reported here show that this may be correct, for when the repository form of Vitamin B_{12} (cyanocobalamin zinc tannate) was used a high percentage of remission of symptoms was obtained in similar cases.

Some Results

The main purpose of this paper is to report some of the results obtained when cyanocobalamin zinc tannate was used in the treatment of patients commonly encountered in private practice. Following are brief histories of these cases and the results obtained.

Case Histories

Case 1: F. M., at 71 year old housewife had been under treatment for heart disease for the past ten years. For the past two years she has been asymptomatic. Suddenly she developed a severe case of anorexia with loss of weight and generalized weakness. Physical examination did not reveal anything to account for the anorexia. She was placed on vitamins and tonics containing Vitamin B_{12} without results. Subsequently, she was given 500 mcg. of cyanocobalamin zinc tannate and another, two weeks later. Her appetite returned completely within one week. She has continued to receive 500 mcg. of cyanocobalamin zinc tannate monthly with no further loss of appetite and her weight is increasing.

Case 2: J. G., a male 71, a retired businessman has been under treatment for arthritis for many years. He also had macrocytic anaemia. Hemaglobin in the low 60 per cents (9.4 gm). Intensive oral and parenteral treatment with many well known standard drug preparations containing Vitamin B_{12} failed to improve the anaemia. After 3 doses of cyanocobalamin zinc tannate of 500 mcg. each, two weeks apart, his hemaglobin rose to 85 per cent (13.3 gm.), This is the highest that it has been in three years. His arthritis is better and he feels better in general. He continues to receive 500 mcg. of cyanocobalamin zinc tannate every four weeks.

Case 3: E. E., is a 54 year old sign painter whose chief complaints were asthenia and fatigue for the past five years. Physical examination was negative except for a low hemaglobin of 70 per cent (11.0 gm.). Oral and parenteral hematinics had no effect on the hemoglobin. When cyanocobalamin zinc tannate was given in addition to the others the hemoglobin rose rapidly above 80 per cent (12.5 gm.) within three weeks. Cyanocobalamin zinc tannate was given every two to four weeks thereafter in 500 mcg. doses. His asthenia has also disappeared.

Case 4: M. G., a 61 year old housewife had a resection of the sigmoid for a carcinoma one year ago. She developed a mild hypertension and anaemia; complained of no "ambition or pep" since the operation. When cyanocobalamin zinc tannate was added to her present medication she felt normal within three weeks. She is no longer anaemic. She receives 500 mcg. of cyanocobalamin zinc tannate every three weeks.

Case 5: M. S., a 51 year old housewife has had hypotension, anaemia, bronchiectases, and asthma for a number of years. Oral and parenteral medications gave only short periods of improvement. With cyanocobalamin zinc tannate added and given in 500 mcg. doses every two to four weeks there has been a decided remission of symptoms in the anaemia, bronchiectasis and asthma.

Case 6: M. P., a 70 years old widow has had a chronic progressive arthritis and anaemia 65 per cent (10.2 gm.) for a number of years. Gets a systemic allergic reaction from most medications. Since she has received 500 mcg. of cyanocobalamin zinc tannate every two to three weeks, the hemaglobin has risen to 82 per cent (12.8 gm.) and the pain and stiffness has lessened considerably and she has tolerated the medication without reaction.

Case 7: R. T., a 41 year old housewife complained of many menapausal symptoms. Hormonal medication and sedatives gave some relief. With the addition of 500 mcg. of cyanocobalamin zinc tannate every three to four weeks there was a decided improvement generally and a remission of all her symptoms.

Case 8: J. D., a 45 year old male alcoholic. He has had terrific "hangovers" following his latest bouts of drinking. He didn't respond to sedatives and vitamins as he had previously. With the addition of cyanocobalamin zinc tannate to the previous medications he was able to return to work within three days instead of three weeks.

Case 9: M. C., a 15 year old girl developed a Bell's Palsy with no other symptoms. No apparent cause could be found for the palsy. After 500 mcg. cyanocobalamin zinc tannate there was beginning improvement the next day with recovery within two weeks. The second dose of cyanocobalamin zinc tannate was given one week following the first.

Case 10: M. B., a 49 year old housewife had psoriasis for many years. There was usually a remission of the lesions during the summer. One year ago she was found to have a secondary anaemia 67 per cent (10.5 gm.). Diet and oral and parenteral medications were ineffective. By adding cyanocobalamin zinc tannate 500 mcg. every three weeks the hemoglobin rose to 85 per cent (13.3 gm.). She claims there is also some improvement in the psoriasis.

Discussion

Some of the above cases were treated with cyanocobalamin zinc tannate alone and others were treated with cyanocobalamin zinc tannate combined with other indicated methods. Only those cases that have been under observation longer than six months were reported. Many of the patients had an anaemia of the "static" type. By this we mean that there was no rise in the hemoglobin level irrespective of the kind of treatment given. Yet every one of these "static" anaemias responded dramatically when cyanocobalamin zinc tannate was added to the regime.

It was further noted that many of these patients had numerous vague and indefinite complaints in just about every part of their body. To our surprise most of these vague symptoms cleared up when the patients were given cyanocobalamin zinc tannate. The best explanation that we can put forth is that they were "hunger starved" for Vitamin B_{12}; in other words the whole body may be affected as in any other constitutional disease. If the body pool of Vitamin B_{12} remains at a subnormal level long enough, then all of the tissues and organs may "slow up". Obviously, it is desirable and important that adequate Vitamin B_{12} levels should be maintained all of the time. Since cyanocobalamin zinc tannate does provide long sustained serum levels and body retention of Vitamin B_{12} it is easy to understand how and why it functioned so effectively. Another interesting observation was that many patients on cyanocobalamin zinc tannate therapy were able to tell by the way they felt when their Vitamin B_{12} availability was diminishing and they would request another dose, usually at from three to five week intervals.

Conclusions

1. Cyanocobalamin zinc tannate is a highly active and notably safe Vitamin B_{12} preparation.

2. A therapeutic dose of cyanocobalamin zinc tannate (500 mcg.) given parenterally will give high sustained serum levels of Vitamin B_{12} for four weeks or more.

3. Cyanocobalamin zinc tannate can be used independently or as an adjunctive to other therapies.

4. Cyanocobalamin zinc tannate provides better and more consistent results than other forms of Vitamin B_{12} previously employed.

5. Cyanocobalamin zinc tannate is very effective in the treatment of a variety of conditions associated with Vitamin B_{12} deficiency commonly seen in private practice.

6. Cyanocobalamin zinc tannate was found effective in a number of cases where oral and parenteral B_{12} of conventional types previously failed.

Summary

Reasons why oral and non-repository forms of vitamin B_{12} are often ineffective are discussed. Clinical experiences with a long acting repository form of B_{12} (cyanocobalamin zinc tannate) which is very effective are presented. A number of cases are reported in which this preparation was used.

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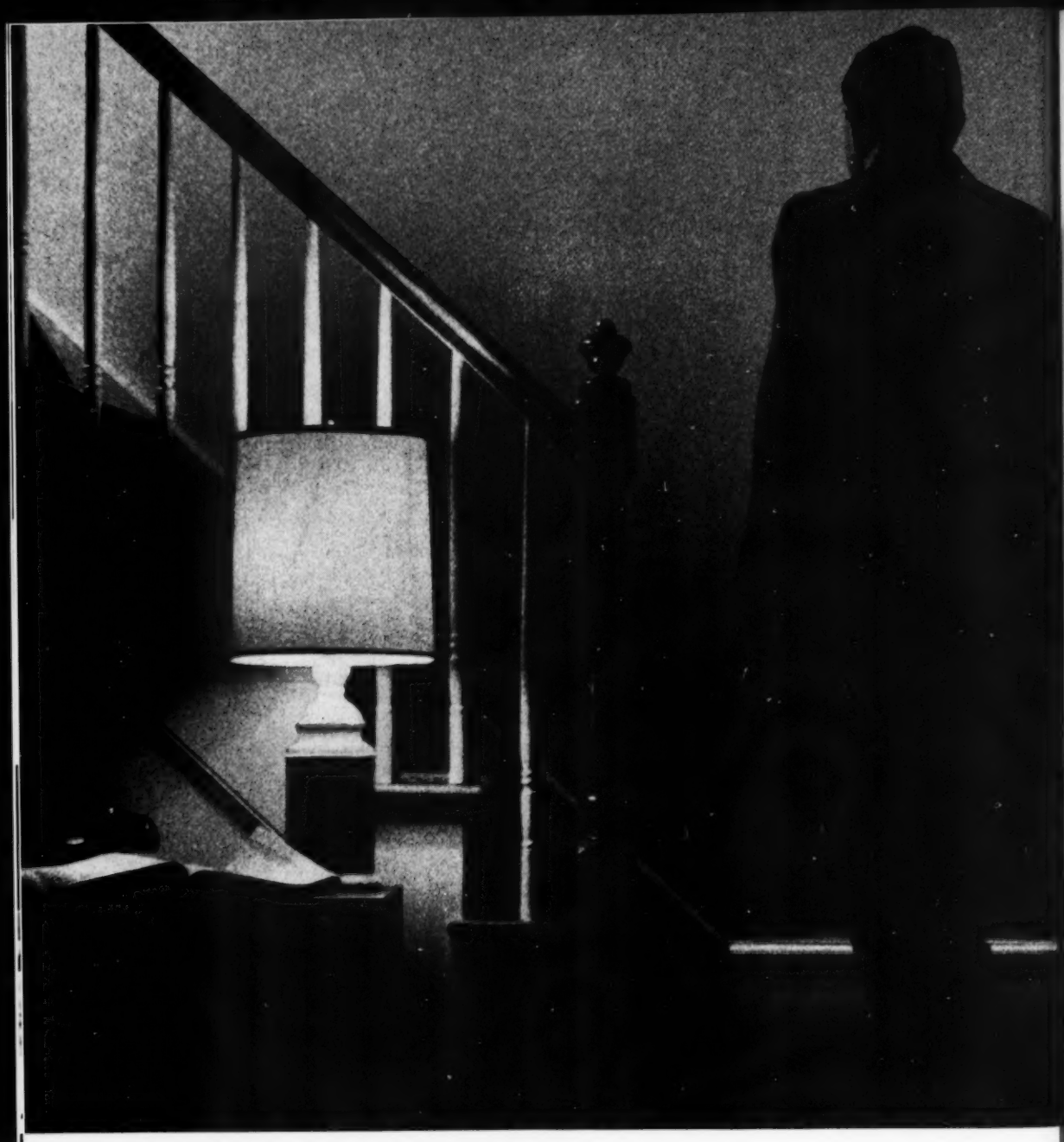

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Clinical Pathological Conference
R. E. Thomason General Hospital, El Paso

September 17, 1959

F. P. Bornstein, M.D. *Editor—Case #1302*

Presentation of case by Solomon Heller, M.D.

History:—Dr. Nathan Kleban

A 28 year old unmarried Latin-American domestic worker was referred to the hospital for admission on May 29, 1959 by the City-County Tuberculosis Control Clinic.

Seven months before admission the patient was subjected to an appendectomy. A greenish-yellow vaginal discharge was noted three months later. Sexual intercourse was denied. Burning discomfort while urinating began one month later, followed shortly by almost constant dull pain in the waist with radiation to both lower abdominal quadrants. Urine was dark.

Constipation had been present for two weeks. Weight was 135 lbs. when illness began. Respiratory symptoms were denied. Admission to the tuberculosis ward of the hospital was said to be prompted by the observation of the Tuberculosis Control Clinic of bilateral apical infiltration on chest film.

Health had been good prior to the present illness except for influenza. Menstrual history was normal.

Family History:

The patient's father died two months before her admission. He was said to have diabetes mellitus, heart trouble and possibly cancer. The mother was said to have had lues. There were two brothers and seven sisters.

Physical Examination:

T. 98.2 P. 88 R. 24 B. P. 118/50 Wt. 101

The patient complained of nausea. No abnormal physical findings were recorded.

Hospital Course:

Multivitamins and ferrous sulfate were prescribed. Sulfamethoxypenthazine (Kynex) was started on the fifth day. Para-amino salicylate and isonicotinic acid hydrazide were begun on the seventh day. Chlorothiazide 500 mg. (Diuril) was given for nine days.

The patient was transferred to the medical ward. A low protein, low salt diet was ordered. Erythromycin was substituted for the sulfamethoxypridazine for six days. Kanamycin 2.0 gms. daily was started on the twenty-second day; then reduced to 1.0 gm. daily two days later.

The patient had few complaints, generally ate fairly well. Castor oil given in preparation for an intra-venous pyelogram was not retained. There was increasing vomiting, leg cramps, lethargy. Brown, granular emesis was noted on the twenty-fourth day. The patient became weak, was unable to get out of bed. Black-green emesis occurred on the twenty-sixth day when retrograde pyelograms were done.

Numbness, difficulty in swallowing, labored respirations and stupor followed. Mandelamine was prescribed. Frost was noted on the face and chest. Respirations ceased and the patient was pronounced dead on the 27th hospital day.

Laboratory Findings:

Blood counts: 6-1-59—Hb. 13.1 gms., Ht. 42, WBC 8,300, 66 Segs., 34 lymphs. 6-22-59 Hb. 10.7 gms., Ht. 36, WBC 6,200, 72 segs., 28 lymphs.

Blood chemistry: 6-1-59—Glucose 95. 6-2-59—Urea Nitrogen 84, Van den Bergh Direct .116, Indirect .435. 6-4-59—Non-protein nitrogen 168,

Urea nitrogen 80. 6-5-59—Non-protein nitrogen 164, Urea nitrogen 80. 6-19-59—Creatinine 11.8, Non-protein nitrogen 280, Urea nitrogen 93. 6-20-59—CO2 capacity 15 mFq/L, Chlorides (as NaCl) 84 mFq/L, Potassium 5.5 mFq/L, Sodium 122 mFq/L, Urea nitrogen 118. 6-23-59—Amylase 330, CO2 capacity 15 mFq/L, Chlorides (as NaCl) 84 mFq/L, Potassium 6.2 mFq/L, Sodium 114 mFq/L.

Sedimentation rate: 6-1-59—42 mm./60 minutes, no correction; 6-19-59—50 mm./60 minutes, correction 42.

Urinalyses: 6-1-59—yellow, turbid, acid, S.G. QNS-albumin 2+, sugar negative, WBC loaded with clumps; 6-2-59—urobilinogen negative; 6-5-59—pale straw, cloudy, alkaline, S.G. 1.006, albumin 2+, sugar negative, loaded with phosphate crystals; 6-10-59—straw cloudy, acid, S.G. 1.005, albumin 3+, sugar negative, WBC 20-30, RBC 30-40, occ. squamous ep. cells; 6-11-59—yellow, cloudy, acid, S.G. 1.006, albumin 2+, sugar negative, acetone negative, diacetic acid negative, WBC gross, RBC gross, occ. ep. cells; 6-19-59—straw, cloudy, acid, S.G. 1.007, albumin 4+, sugar negative, urobilinogen negative, WBC 40-50, RBC 25-35, few squamous ep. cells, casts negative, bacteria negative; 6-21-59—yellow, turbid, alkaline, S.G. 1.014, albumin 3+, WBC loaded, with clumps, RBC 8-10, few squamous ep. cells, few motile bacteria.

Urine, direct smear: 6-10-59—many WBC, no bacteria seen.

Urine for Bence-Jones Prot: 6-16-59—negative.

Urine culture and sensitivity: 6-5-59—Gram. ng. rods, Sens. Kanamycin, Neomycin.

Serology: 5-30-59—Negative.

Sputum: 6-1-59—No A.F.B. seen, culture negative; 6-8-59—No A.F.B. seen; 6-12-59—No A.-F.B. seen.

P.S.P.: 6-11-59—

1st Vol. 105 cc	% dye exc. 2.0%
2nd Vol. 72 cc	1.2%
177 cc	3.2%

A.S.O. Titer: 6-19-59—166 Todd units.

C. React: Proteins: 6-18-59—Pos. (+ 1)

X-rays: 6-2-59 — Gallbladder, Chest — Conclusions: Healthy chest, no evidence of active pulmonary tuberculosis. Normally concentrating and poorly contracting gallbladder. No evidence of calculi.

6-12-59 — P. A. Chest — Conclusions: Healthy chest.

6-15-59—Intravenous Pyelogram: Survey film of the abdomen reveals the kidneys to be normal in size, shape, and position. The psoas shadows and properitoneal fat lines are present bilaterally. No significant intra-abdominal opacities can be identified. The lumbar spine and pelvis appear natural. Following the intravenous administration of hypaque, there is non-visualization at serial-graphic observations up to 15 minutes. Re-examination following dehydration of the patient is recommended for further evaluation. Conclusion: Non-visualization, possibly on a technical factor. Re-examination for confirmation is recommended.

6-24-59—Retrograde Pyelogram: Survey film of the abdomen reveals an opaque catheter lying in the upper third of the left ureter. Following the instillation of opaque media, there are findings consistent with an advanced hydronephrosis. There is some irregularity of the visualized ureters most probably secondary to a ureteritis with accompanying hydronephrosis. The bladder contour is irregular consistent with an accompanying cystitis. Conclusion: Findings consistent with obstructive uropathy on the left.

Serum protein electrophoretic pattern 6-18-59:

			Norm.
Albumin		33.8%	56±8
Globulin	Alpha 1	8.1	6±2
	Alpha 2	20.7	10±5
	Beta	15.9	13±4
	Gamma	21.5	15±5

Clinical Discussion—Dr. Solomon Heller:

This is the case of a 28-year-old woman, who was admitted with minimal pulmonary tuberculosis, and proceeded to develop anemia and died of uremia in 27 days, unrelated, apparently to the pulmonary process. There is a series of red herrings in the beginning, I believe, which make you wonder. Apparently seven months before admission the patient was subjected to an appendectomy, we don't know whether this was a ruptured appendix, whether there was any difficulty en-

countered at the time or whether it was a more or less routine procedure. In view of the subsequent course you wonder whether there wasn't some injury to the ureter. A greenish vaginal discharge was noted three months later, or roughly four months prior to admission.

The lady denied having had intercourse but there is the possibility of a granulomatous pelvic inflammatory disease which would have no relation to intercourse. Then appeared burning urination a month later or three months prior to admission, and again dull pain in the waist, with radiation to both lower quadrants, and a dark urine, which possibly means hematuria. Again you wonder whether there isn't besides the presence of dysuria, hematuria or at least a dark urine, the possibility of inflammation in the pelvis.

Weight Loss

There was a weight loss of roughly 34 pounds from the time the patient had last noted a weight of 135 pounds to the time of admission when she weighed 101 pounds. On examination at the time of admission the blood pressure was normal and remained so throughout the course of hospitalization. The temperature was also normal on admission which makes it difficult to imagine a chronic inflammatory disease, although it can happen. Only twice during the patient's hospitalization was there a spike in temperature up to 99.8 and 99.6.

There are a few comments I would like to make about the medications. It seems to me that the dosages of these medications are somewhat out of line with the patient's status of azotemia and later uremia. It is true that they were treating what appeared to be a gram-negative organism, possibly an infectious process in the kidneys, but particularly Panamycin which is a nephrotoxic agent, should have been used a little bit more judiciously. The patient from the time of admission had a rather progressively rapid downhill course, dying of uremia.

The initial hemoglobin of 13.0 grams is strikingly normal for someone who may or may not have been chronically uremic. Interestingly enough the hemoglobin of June 22 was 10.7. This is much more compatible with a patient chronically uremic; they generally run eight to 10 grams. Possibly the patient had been vomiting, and was dehydrated. However, according to the chart the patient ate well, at least initially.

Normal Glucose

Another interesting item is the normal glucose from the differential diagnostic standpoint, at least on the first of June. Of course there is the remarkable rise in the urea nitrogen and NPN subsequently. The amylase of 330 is somewhat puzzling. I don't know why it was ordered. There is the possibility of course that, as occurs occasionally in uremia, there is involvement of the pancreas. As I recollect there are certain characteristic changes in the pancreas of uremics some of which are of obstructive nature.

The sedimentation rate was elevated on both occasions which would speak for some active process. All of the urines show anywhere from eight to 10 actual clumps composed of red cells and white cells as well as albuminuria from two to four plus. I couldn't tell from this whether any of these urines were catheterized.

One other point I would like to ask is whether a pelvic examination was done?

Dr. Pablo Ayub:

I think it says here she denied any sexual intercourse. Probably for that reason it was not done.

Dr. Heller:

There are a number of cultural reports here, and I believe one of these was a urine which was negative for acid-fast organisms. The PSP was markedly decreased as you would expect. I would wonder very seriously about the rationale of the use of the intravenous pyelogram with the highly elevated NPN. The retrograde pyelogram and the chest X-ray I would like to have interpreted for me.

X-Ray Discussion—Dr. Charles C. McVaugh:

The admission chest is essentially negative. There is no evidence of uremia in the chest X-ray. Certainly uremia is a contra-indication to intravenous pyelography and the house staff has learned that now. In the presence of an elevated NPN there is nothing to be gained by requesting an IVP because they rarely if ever visualize, certainly with an NPN elevated as highly as this one was.

The left retrograde pyelogram reveals a rather pronounced left hydronephrosis, the left ureter is not remarkably dilated, I believe there is some obstruction at the pelvic-ureteral junction, the right urinary tract is filled, the right kidney appears normal.

Dr. Ayub:

Did they put a catheter in the right kidney?

Dr. McVaugh:

There is no catheter that I can visualize.

Dr. Heller:

Does anyone know if they attempted to put a catheter on the right?

Dr. McVaugh:

There is rather a pronounced irregularity of the bladder but it is incompletely filled, not completely visualized and I believe there is probably extrinsic involvement of the bladder by pelvic inflammatory disease. Incidentally I see linear densities of gas in the right upper quadrant which may be outside the gut. This may represent free air beneath the liver or around the gallbladder. It is very difficult to tell. I don't believe it's colon or small bowel. However, I see no other evidence indicating peritonitis or acites but these linear densities are very suspicious of free air in the abdomen.

Dr. Bornstein:

This girl was admitted supposedly for an apical lesion. Is there any evidence of pulmonary tuberculosis?

Dr. McVaugh:

No, I said the chest was essentially negative unless there is a tiny lesion behind the clavicle, which I can't see. I see no evidence of an active lesion.

Dr. Francisco Licon:

What is your diagnosis, Doctor?

Dr. Bornstein:

What positive lesions do we have?

Dr. McVaugh:

A left hydronephrosis which I think is due to an obstruction of the pelvic-ureteral junction. I believe there is extrinsic involvement of the bladder, although it is difficult to tell because of the inadequate filling, and possibly a ruptured hollow viscus.

Dr. Heller:

I frankly wonder on looking at this X-ray whether there might not also be some involvement of the right kidney. There is a very suggestive shadow here. It may be gas, of course, or an accumulation of feces but it is very suggestive that a similar process is present on the right.

We are to assume that there is an obstructive process at the uretero-pelvic junction of the left kidney. This is a place for a congenital anomaly, possibly anomalous vessels with secondary obstruction and secondary infection which would make a very nice picture.

However, there must have been something wrong with the other kidney as well, to drive the patient progressively into azotemia. Given a patient in uremia, we have a rather massive differential diagnosis, possibly particularly in women this age, a young individual, glomerulonephritis would be one of the first things we suspect.

Secondary Phenomena

However, that is not an obstructive process unless there are secondary phenomena. Other diagnoses would be systemic lupus, a Kimmelsteil-Wilson's disease would be unlikely particularly with a hydronephrotic process. It is amazing to me that a kidney can be this far gone and unilateral, although I think it is bilateral, and maintain normal blood pressure, especially if there is a chronic infection which at least was very severe in the end phase, although there was no fever.

Renal Tuberculosis

I think in spite of the fact that she had no pulmonary lesion you have to consider very strongly a renal tuberculosis, particularly in this region of the United States. This disease generally results in activation of a focus from some other primary site. Ten percent of individuals with known tuberculosis will develop renal tuberculosis supposedly somewhere around 10 years after the primary infection.

They are very prone to develop ureteral obstruction and have a destructive process with hydronephrosis, leaving a shell of the kidney with complete loss of function. Apparently on X-ray there is no direct evidence that would confirm the diagnosis of tuberculosis. One other disease particularly in the face of an obstructive uropathy

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*Thompson, R. E., and Hecht, R. A.: Am. J. Clin. Nutrition 7:311-317 (May-June) 1959.

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which you would have to consider is a necrotizing papillitis.

Necrotizing Papillitis

Necrotizing papillitis is a disease present in diabetics somewhere about 62 per cent, a lesser number is found with obstructive uropathy, and approximately 10 per cent with neither. This woman had a good chance of pyelonephritis and she has what appears to be an obstructive process with hydronephrosis, and possibly this may have been the final complication that killed the patient. Generally, though, there is a rather obvious septicemia associated with this condition, although an overwhelming infection with no fever may be present.

Other Possibilities

Other etiological possibilities have to be considered for this obstruction. There may be a metastatic tumor or an inflammatory process arising from the pelvis, although it is rather high for a pelvic inflammatory disease. A lymphoma might infiltrate the ureters from the retro-peritoneal space and cause obstruction. For this there was no evidence, not hepato and splenomegaly. Periureteric fibrosis, due perhaps to some other inflammatory process or even primary, would lead to obstruction and secondary inflammation.

Quite frankly the most obvious solution would be to postulate a congenital anomaly with aberrant vessel which obstructed one, and I think actually both kidneys with super-infection by coliform organisms with a rather progressive destructive pyelonephritis leading to uremia. I cannot rule out somewhere that there may have been a superimposed renal chronic papillitis or possibly even tuberculosis.

Dr. Ayub:

There are very few conditions where a progressive uremia could exist with a normal blood pressure. They are tuberculosis, amyloidosis, multiple myeloma, bacterial endocarditis and polycystic kidneys. Nearly all other renal lesions affect the vascular supply of the kidney and this in turn produces secondary hypertension. The patient had persistent pyuria and we made all possible effort to demonstrate tuberculosis. The possibility of a polycystic kidney is also present. There was no history of serious infection and no signs of liver damage which would have suggested amyloidosis. The Bence-Jones protein was normal, which speaks against multiple myeloma although it does not rule it out.

Dr. H. M. Gibson:

I think this patient had a chronic uremia, possibly with an exacerbation of some sort. I think we have to assume that this patient had a chronic renal disease. I think the right kidney shadow is enlarged. The obstruction of the left side is distinctly uretero-pelvic. There is nothing in the lower ureter here which is causing obstruction, at least on the visible kidney. I suspect that this patient had bilateral obstructions of both kidneys at the uretero-pelvic junction, with uremia, and that the situation simply gradually became exacerbated and one injury piled on top of the other. These people look washed out, they look sick, they look a lot like tuberculous patients. Looking at the X-rays on the left side there is some faint moth-eaten appearance of the calyces.

Slightly Compatible

Although this is not at all typical of tuberculosis, it may be slightly compatible with the possibility of tuberculosis but not at all diagnostic. Now, you gentlemen on the house staff, let me make a couple of remarks about intravenous pyelograms: you do not ever order an intravenous pyelogram on a patient who appears debilitated or an elderly patient without getting a urea nitrogen. If the urea nitrogen is elevated, say 25 or more, NEVER order an intravenous pyelogram for two reasons. First of all, you will not get a picture. Second, the dye is excreted in the renal tubules, and this dye produces distinct and definite damage to the renal tubules.

It is temporary in a normal kidney. You do not believe this? Get an NPN and follow the patient along, give the patient an intravenous pyelogram and then follow the urea nitrogen and NPN's. It is entirely possible that the intravenous pyelogram aggravated the disease of this patient. I cannot over-emphasize the point, do not ever order an intravenous pyelogram on a patient with a urea nitrogen over 25.

So my impression is that this patient simply had a bilateral congenital obstructive uropathy and that she died a uremic death. I would like

to make one point as far as polycystic kidneys are concerned—I am sorry, but the vast and overwhelming majority of polycystic kidneys do have hypertension.

Dr. Ayub:

But a normal blood pressure can be associated with polycystic kidneys.

Dr. Gibson:

Well, anything can be. You can have possibly a classic Goldblatt kidney without hypertension, but for all practical purposes polycystic kidneys in the overwhelming majority of cases are associated with hypertension.

Dr. Licon:

I would like to ask a question of one of the experts. It states that there are no abnormal physical findings. This patient is not particularly obese. She had these fairly large looking kidneys. I was wondering whether they made any examination for palpation of the kidneys, given the urinary history and what not, and I would like to ask Dr. Gibson how large does a kidney have to be before you can palpate it.

Dr. Bornstein:

I would think it depends on the size of the panniculus adiposus. I am very anxious to hear from Dr. Postlewaite especially because the City-County Health Department was so very positive about this patient having tuberculosis, and here everybody made rather light of this observation.

Dr. Jack C. Postlewaite:

Now the business of tuberculosis. We can work backwards on this. Apparently it is backwards, because at the moment I see some density in the right apex here, several areas of density suggestive of something that's been going on. One of them is a little bit soft. Has the radiologist left yet? In spite of the fact that this is spine technique here is a residual in the right apex, and as a result there is something suggestive, and you can see how the tuberculosis viewers could have picked it up on those small films.

I saw this patient on the tuberculosis ward. When we transferred her, this was a very ill woman, even as early as this protocol picks up here, which is about a month in the hospital. Dr. Licon struck a very good note there. To say the examination was negative is stretching it a little bit because this woman had liver tests, she was jaundiced, she had a weird looking color and she was definitely sick. Dr. Gibson gave us a clue. This girl was uremic and had been for a long time.

Chronic Renal Disease

This was nothing that came on rapidly. The deterioration of this woman isn't unduly rapid for 30 days of hospital, but I think it is correct to assume that this woman had chronic renal disease, probably obstructive. Then here she developed hematuria.

In the beginning she had a negative catheterized urine and later on she had blood in the urine.

Now you must add to your differential diagnosis that she had a malignancy, that she had tuberculosis, two of the most common causes of hematuria, and that she may have developed an acute disease superimposed upon the chronic one such as perhaps an acute glomerulonephritis superimposed on a chronic hydronephrosis, obstructive, and ascending pyelonephritis.

Drug Therapy

Dr. Gibson talked about the IVP dye causing trouble. I would like to note that in drug therapy, as already mentioned by Dr. Heller, she was getting good doses of anti-tuberculosis drugs. You will find that these doses have been for management, and with an obstructive lesion you retain large quantities of PAS and I N H and you develop a nephrotoxic problem. She also must have received streptomycin. So she deteriorated rapidly on the tuberculosis ward and I regret that we kept her that long, although tuberculosis is still a good possibility.

Dr. Ayub:

How about the lack of elevated blood pressure on your diagnosis of glomerulonephritis?

Dr. Postlewaite:

There is something to suggest a salt-losing kidney which is a terminal kidney.

Dr. William R. Gaddis:

I feel bound to remark on one thing in this patient's history, that is that this patient under-

went an appendectomy seven months prior to her being admitted to the hospital. One must take into consideration the fact that this patient may have had abdominal findings and/or symptoms of this present disease as early as seven months prior to her admission to this hospital.

We do not know what was found on surgery, primarily because we have no record of that on this side of the river. She was apparently admitted to a hospital in Juarez and subjected to the appendectomy over there. I am assuming that this patient had abdominal findings which suggested the necessity for surgery, which was done. All of us who see patients know that the findings with urinary tract disease can be very confusing; and on occasion patients have been subjected to appendectomies in our hospitals for urinary tract disease.

It is quite possible and well in keeping with this patient's record that she was subjected to the appendectomy and probably had a normal appendix or at least that is what Dr. Bornstein would report. That merely marked the end phase of the beginning of the end as far as her condition was concerned.

Clinical Diagnosis: Chronic pyelonephritis with uremia.

Dr. Heller's Diagnosis:

1. Congenital anomaly of both kidneys with obstruction and superimposed destructive pyelonephritis.
2. Renal tuberculosis?

Pathological Diagnosis:

1. Bilateral caseous necrotizing pyelonephritis compatible with tuberculosis.
2. Bilateral pulmonary apical tuberculosis.

Pathological Discussion—Dr. Bornstein:

This was an emaciated woman, there was nothing unusual externally except the appendectomy scar. The heart was not remarkable. The lungs were pinkish-grey and spongy. On careful examination small indurated depressed areas were found in each apex with stellate scars on the dome of the pleura on the parietal side. On sectioning in these areas each lung in the apex showed circumscribed regions, blackish-brown in color, very firm, with areas of caseation necrosis. Each of these lesions measured three cm in greatest diameter. The remainder of the lungs were free of lesions.

The only other lesions were found in the kidneys. Both kidneys had a fairly similar appearance. They were somewhat large, flabby, and had depressed lesions in the cortex. On opening both kidneys again had approximately the same appearance. There were large, distended renal pelves filled with a pale grey, rice water-like fluid, distended renal papillae with deposition of a greenish necrotic material (fig. 1). I made a preliminary gross diagnosis of caseous tuberculosis of both apices and a bilateral renal far advanced tuberculosis.

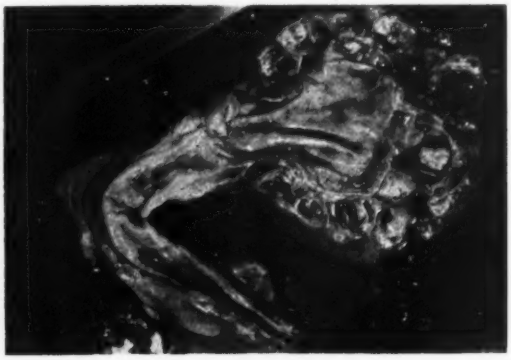

Figure 1

Lymphocitic Infiltration

On microscopic examination things became a little more complicated. The sections of the lung showed foci of lymphocytic infiltration and very marked fibrosis and that is about all. There were no acid-fast bacilli demonstrable in the lung. The kidneys were even more baffling. This represents the main type of lesion in the kidneys (fig. 2). There were tremendous areas of necrosis, abscess-like, with a rather bland margin without any good reaction around it.

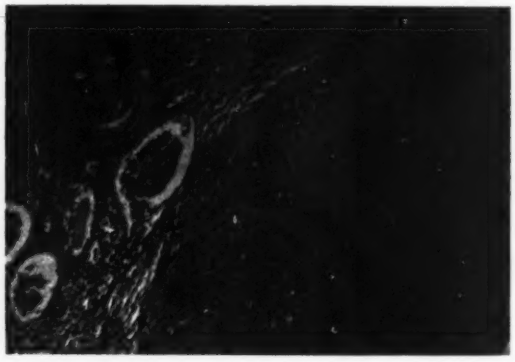

Figure 2

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Very occasionally one sees necrosis and a few epithelioid cells, very occasionally necrosis with a few Langhans' giant cells. Now this, under ordinary circumstances and especially in view of the fact that we never found acid-fast organisms, I would never have dared to call tuberculosis, except for one thing—this patient had received anti-tuberculosis chemotherapy; and as Dr. Gibson, who has given me a number of these kidneys on surgery, can well confirm, the morphology, the histopathology of tuberculosis of the kidney treated with antibiotics and anti-tuberculosis drugs, becomes very, very atypical.

If, then, we have pulmonary foci and a gross lesion of bilateral saccular hydronephrosis with caseation necrosis, then I feel that the most reasonable diagnosis here is renal tuberculosis, and I would say that the chemotherapy is responsible for the fact that we did not find any acid-fast organisms.

Question:

Dr. Bornstein, did you see any obstruction at the uretero-pelvic junction?

Dr. Bornstein:

No. I saw some edema in the ureter, but the main causes of obstruction are the big, saccular abscesses filled with caseous material which represent a rather characteristic form of renal tuberculosis. There are actually two types of renal tuberculosis, the type where you get the large, saccular structures, and the one where they get the small granulomas.

Dr. Ayub:

Would you expect to find bacteria in the cheesy material that you could inject into guinea pigs?

Dr. Bornstein:

We might have found them if we had tried. There are other elements superimposed in this patient because usually renal tuberculosis does not go down hill that fast. Admittedly this patient came in in uremia and got some rather harsh treatment for her kidneys, but I think some secondary organisms are involved here, but I believe that the basic lesion here was a tuberculosis lesion.

Dr. Kleban:

She didn't receive anti-tuberculosis chemotherapy for over a week. Possibly it was begun on the seventh day and a few days later, when Jack saw her, she was transferred to medicine and that was stopped. I doubt if she received it for more than a week.

Social Security Footnotes

There is not an unlimited number of ways for Social Security to expand. Medical care is one of the few areas not covered by "social insurance," and the present framework of the Social Security Act is adequate to cover socialized medicine by means of a few amendments. The Disability Insurance "Trust" Fund could be changed into a Health Insurance "Trust" Fund by the stroke of a pen. Taxes could be increased. A new title could be added to the law and the private practice of medicine could be virtually destroyed.

In one way or another, on one excuse or another, social security tax payments are being boosted every year or two instead of at five-year intervals as originally planned. In 1954, the base was rased from $3600 to $4200. In 1956, the tax rate was increased. Now, Rep. Kean wants to raise the base rate from $4200 to $4800 beginning in 1959. Then in 1960, the tax rate is scheduled to increase ½ percent for both employee and employer, and ¾ percent for the self-employed. There is no way of knowing just how expensive social security "insurance" is actually going to be.

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IN THIS ISSUE

The President's Page
The Physician and Community Leadership ..... **Page 755**

Lead Exposure — A Common Cause of Disease ..... **Page 757**

Southwest OB-GYN Society Annual Meeting in Palm Springs
Picture Report ..... **Page 763**

Clinical Experiences With A New Long Acting Vitamin B_{12} ..... **Page 767**

Clinical Pathological Conference, R. E. Thomason
General Hospital, El Paso ..... **Page 771**

COMPLETE CONTENTS ON PAGE 746

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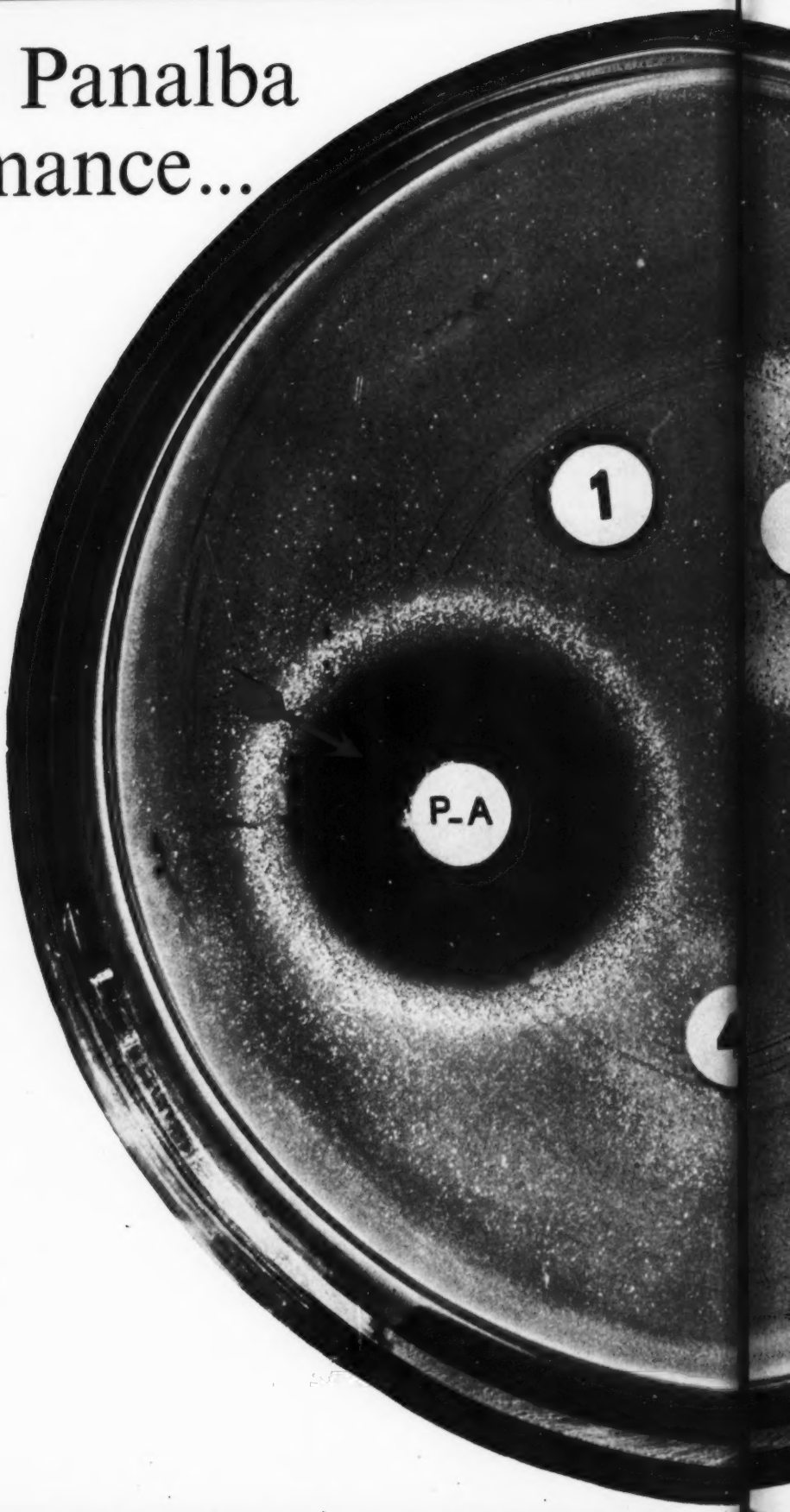

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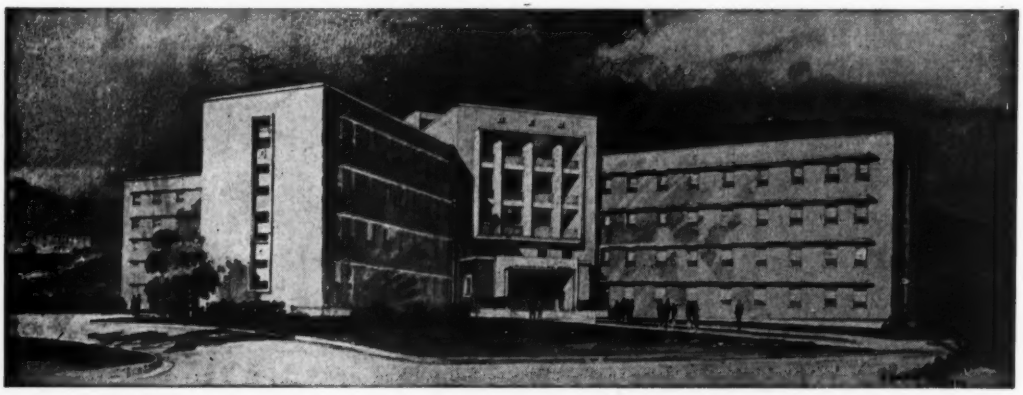

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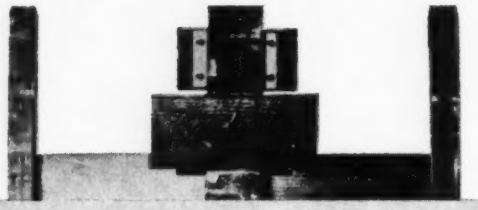

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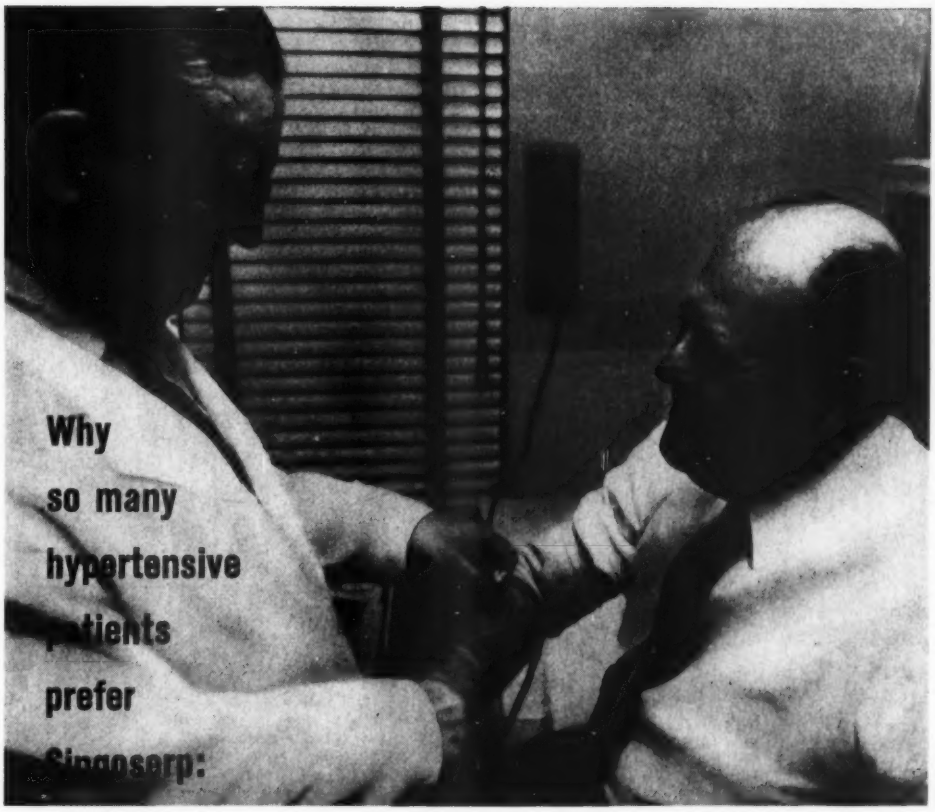

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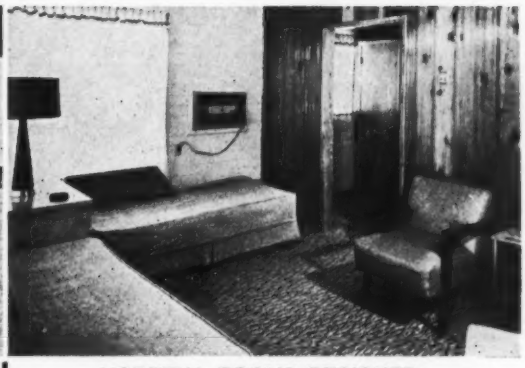

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1. Elsdon—Dew, R.: S. Afr. Med. J. 32:89 (Jan., 1958)
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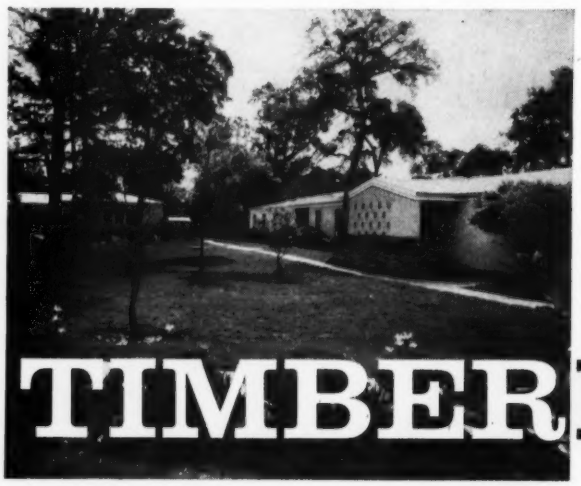

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